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BRIEF REPORT

Smoking and bladder cancer risk in Blacks and Whites in the United States

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A population-based case-control study of bladder cancer (2,982 cases and 5,782 controls) conducted in 10 areas of the United States examined the effect of smoking as a risk factor among Blacks and Whites, after adjustment for occupation and other potential confounders. Although the overall risk for smoking was slightly higher in Blacks than Whites (relative risk = 2.7 and 2.2, respectively), this difference was not statistically significant. Estimation of risk by dose and currency of exposure revealed no consistent racial disparities in smoking-related risks. Race-specific, attributable risk estimates indicated that nearly half of bladder cancers among both Blacks and Whites could have been prevented by elimination of smoking.

Key words: Blacks, bladder cancer, case-control study, epidemiology, race, smoking, United States, Whites.

Introduction

The incidence of bladder cancer is now 18.2 per 100,000 person-years (PY) in the United States White population and 9.8 in the Black population.[1] Relative survival five years after diagnosis is 79 percent and 58 percent, respectively. Incidence and survival combine to create the same mortality rate in the two groups, 3.3 per 100,000 PYs.

In 1988, we assessed how much of the apparent racial disparity in incidence could be ascribed to different exposure patterns based on data from a case-control study of bladder cancer. We identified 2,982 incident cases from cancer registries in the Surveillance, Epidemiology, and End Results (SEER) program and 5,782 from the general population resident in the areas covered by the cancer registries.[2] We purposely did not stratify on race in the selection of study subjects, in order to estimate the effect of race with and without adjustment for smoking, occupation, and other risk factors. We found fewer Black subjects had ever smoked but, among those who had ever smoked, fewer had stopped smoking at least one year before the study.[3] Black subjects were slightly less likely to have smoked filtered cigarettes. Differences in exposure patterns were not great enough to explain the overall excess incidence among Whites, which was reduced from 90 percent to 60 percent by adjustment for cigarette smoking and occupation.

One plausible explanation for the residual racial

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disparity is less frequent diagnosis of early-stage lesions in Blacks. In our data, the marked racial disparity in stage at diagnosis persisted even after adjustment for known risk factors. Carcinoma *in situ* or limited to the mucosa was found about five times as often in Whites; cancers invading the submucosa, 2.7 times as often; those invading the musculature, about as often; and those extending beyond the bladder, less often.[3]

Another explanation we examined was that smoking or some other risk factor exerted a weaker effect in Blacks. A summary measure, the overall relative risk (RR) for any history of smoking, was 2.7 in Blacks and 2.2 in Whites, a difference that was not statistically significant and in the reverse direction of the hypothesis.[3] Two subsequent reports have suggested that the risk of bladder cancer associated with cigarette smoking may differ in Blacks and Whites. Burns and Swanson[4] compared bladder cancer patients with colorectal cancer patients and found smoking-related RRs to be higher among Black men and Black women than among White men and White women. Harris *et al*[5] compared bladder cancer patients with patients hospitalized for a variety of conditions (benign and malignant neoplasms accounting for 32 percent) and found risk ratios higher in White men than Black men.

We report here on details of the association between smoking and bladder cancer among Black and White subjects. In particular, we examine the racial differences in the RRs according to dose and currency of cigarette exposure, specific for sex, and we compare Black and White subjects with a common reference group so that the effects of race and smoking can be compared with each other.

Materials and methods

The study methods are presented in detail elsewhere.[2] The source population for cases and controls included five states and five metropolitan areas in the US National Cancer Institute's SEER network of cancer registries in 1978. Incident cases of bladder cancer were identified by the registries. A sample of the source population aged 21 to 64 years was obtained by random-digit telephone dialing, with a response rate of 88 percent. A sample of the source population aged 65 to 84 years was drawn from the Health Care Financing Administration files, with a coverage rate of 98 percent. The sampling of controls was stratified by age, sex, and area, but not race. Personal interviews lasting about one hour were conducted in the home. Seventy-three percent of cases and 83 percent of controls were interviewed. We obtained detailed histories of tobacco use, occupation, residence, and use of artificial sweeteners, as well as demographic data and less detailed data on a variety of other exposures.

Findings are given elsewhere on the smoking risks[6] and occupational risks.[7-9] In this analysis, we categorized smoking history as a combination of average dose (< 20, 20+ cigarettes per day) and current smoking status. Of the total 2,982 interviewed cases, we excluded 47 who classified their race as other than Black or White, as well as eight Black and 248 White cases with some data missing, leaving 121 Black and 2,558 White cases in the analysis. Of the total 5,782 interviewed controls, we excluded 121 who classified their race as other than Black or White, and 20 Black and 321 White controls with some data missing, leaving 383 Black and 4,937 White controls in the analysis.

In this analysis, we used a yes/no indicator of occupational risk, namely, whether the subject ever worked (i) in a job for which we identified a 50 percent increase in risk, or a consistent duration effect; or (ii) in a job found to have increased risk in other studies and at least a 30 percent increase in risk in this study.

We estimated the RR by fitting logistic regression models[10] that included indicator terms for age (< 65, 65-74, 75-84), sex, occupational risk (yes/no), and history of smoking pipes or cigars (yes/no). Addition to the model of terms for coffee consumption did not alter the findings. We assessed the statistical significance of the racial difference in RRs by including a term for interaction in the model. We estimated attributable proportions using the method given by Rothman.[10]

Results

Table 1 shows the higher prevalence of current and former smoking among cases than controls, and among men than women. Among the male controls, fewer Blacks than Whites had ever smoked (63 percent *cf* 69 percent), but more Blacks than Whites were current smokers (31 percent *cf* 27 percent). Among the female controls, more Blacks than Whites had ever smoked (40 percent *cf* 35 percent), and more Blacks than Whites were current smokers (31 percent *cf* 21 percent).

The estimated RRs for cigarette smoking were higher among Black subjects than White at some of the exposure levels (Table 2), but the pattern was not strong or consistent. The differences in relative risks between Blacks and Whites were not significant in men ($P = 0.76$), women ($P = 0.67$), or overall ($P = 0.85$).

Table 3 illustrates the combined impact of race and cigarette smoking, adjusted for sex, age, and pipe and cigar smoking, and permits the comparison of additions to risk. The baseline or unexposed rate is that of Black nonsmokers. The data show generally similar

Table 1. Percentage distribution by smoking history among cases and controls, according to sex and race

Cigarettes	Male				Female			
	Black		White		Black		White	
	Cases	Controls	Cases	Controls	Cases	Controls	Cases	Controls
Never	18	37	17	31	39	60	46	65
Ever	82	63	83	69	61	40	54	35
Ex smoker								
< 20/day	12	16	10	13	15	7	11	8
20+/day	15	16	30	28	9	3	5	6
Current smoker								
< 20/day	19	17	8	7	27	24	15	11
20-40/day	30	13	27	16	9	7	19	9
40+/day	6	1	8	4	0	0	3	1
Total	100	100	100	100	100	100	100	100
No. of subjects	88	277	1,925	3,642	33	106	633	1,295

Table 2. Relative risks for bladder cancer[a] (and 95% confidence interval) according to smoking history, by sex and race

Cigarettes	Male		Female	
	Black	White	Black	White
Never	1.0	1.0	1.0	1.0
Former				
< 20/day	1.6 (0.70-3.9)	1.3 (1.1-1.6)	3.6 (0.96-13)	2.0 (1.4-2.7)
20+/day	1.8 (0.79-4.1)	1.9 (1.6-2.2)	5.0 (0.89-28)	1.3 (0.86-2.0)
Current				
< 20/day	2.2 (1.0-4.8)	2.1 (1.7-2.6)	1.7 (0.63-4.7)	2.0 (1.5-2.7)
20+/day	4.5 (2.1-9.3)	3.0 (2.6-3.6)	2.1 (0.44-10)	3.1 (2.4-4.2)

[a] Adjusted for age, geographic area, occupational risk, and pipe or cigar use.

Table 3. Relative risks for bladder cancer[a] (and 95% confidence interval) according to race and cigarette smoking history, with a combined reference rate

Cigarettes	Black	White
Never smoked	1.0 (ref.)	1.7 (1.1-2.5)
Former smoker		
< 20/day	1.9 (0.9-3.7)	2.4 (1.6-3.7)
20+/day	1.9 (1.0-3.8)	3.1 (2.0-4.6)
Current smoker		
< 20/day	2.1 (1.1-3.8)	3.5 (2.3-5.4)
20+/day	4.2 (2.3-7.5)	5.2 (3.4-7.8)

[a] Adjusted for age, sex, occupational risk, and pipe or cigar use.

effects of smoking in Blacks and Whites. A clear racial gap with higher risk in Whites than Blacks also appears at all levels of smoking.

Table 4 shows the incidence of bladder cancer in the source populations at the time of the study and the attributable proportions. Smoking accounted for over 40 percent of bladder cancer among White men, Black men, and Black women, and for 31 percent among White women. One gauge of the racial disparity not attributable to the different smoking patterns is the imputed incidence among people who never smoked. By subtracting the proportion attributable to smoking, one can infer that 16 cases per 100,000 PYs occurred among the White men who never smoked, and 7.9 among the Black men who never smoked. That is, the baseline bladder cancer risk is lower in Blacks than in Whites.

Table 4. Average 1978 incidence of bladder cancer and proportion attributable to smoking, by sex and race

	Male		Female	
	Black	White	Black	White
Average incidence[a]	15.1	29.7	5.2	7.7
Attributable proportion[b]	48%	46%	42%	31%

[a] Cases per 100,000 person-years in 1978, age-adjusted to the 1970 US Standard Population.

[b] Proportion of bladder cancer cases attributable to current and former cigarette smoking.

Discussion

There are several reasons to investigate whether cigarette smoking affects bladder cancer risk differently in Blacks and Whites. There are familial, genetic and, potentially, racial differences in the metabolism of some carcinogens. Further, bladder cancer incidence-rates differ in Blacks and Whites. Therefore, reports such as those of Harris *et al*[5] and Burns and Swanson[4] add to our understanding of racial differences in cancer risk. In such investigations, we believe it is useful to estimate absolute as well as RR measures to perceive the magnitude of racial differences. Employing such an analysis, we found the effects of smoking to differ little between Blacks and Whites. In particular, the smoking-associated RRs were higher for Blacks in several but not all smoking levels. At all levels of smoking, Whites had higher absolute risks.

Several aspects of the study strengthen the inferences drawn from these data. The study base was the general population. We obtained detailed data on smoking and other risk factors. The size of the study, the largest to date, allowed precision in dose-specific estimates, except within the small subgroups, such as Black women.

This study also clearly demonstrates that Blacks as well as Whites have suffered substantial smoking-associated excesses in bladder cancer incidence. Nearly half of bladder cancers among both Blacks and Whites could have been prevented by the elimination of smoking. Large public health benefits of reduced smoking would accrue to Black and White populations.

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